4 **Xiao Z**, Theroux P. Clopidogrel inhibits platelet-leukocyte interactions and thrombin receptor agonist peptide-induced platelet activation in patients with an acute coronary syndrome. *J Am Coll Cardiol* 2004;**43**:1982–8.

5 **Quinn MJ**, Bhatt DL, Zidar F, *et al*. Effect of clopidogrel pretreatment on inflammatory marker expression in patients undergoing percutaneous coronary intervention. *Am J Cardiol* 2004;**93**:679–84.

IMAGES IN CARDIOLOGY

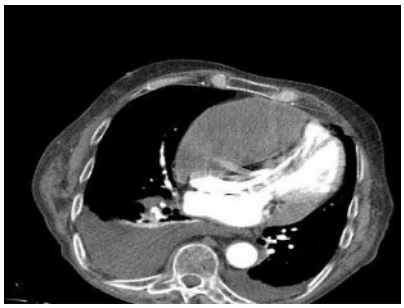
doi: 10.1136/hrt.2005.078063

Cardiac compression from a rheumatoid pericardial mass

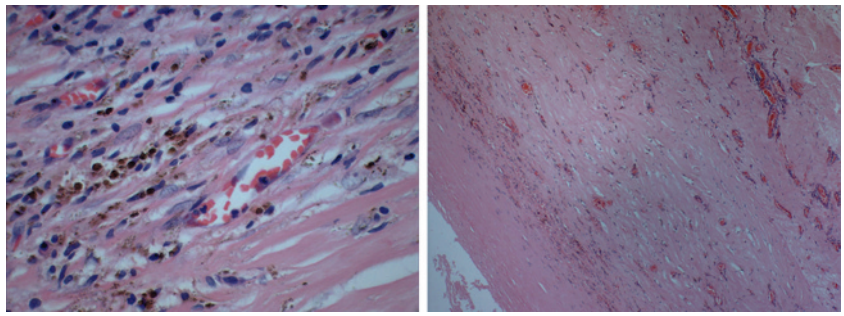
A 75-year-old man presented with progressive dyspnoea. There was a 24-year history of severe deforming rheumatoid arthritis for which he had required a number of disease modifying therapies. At the time of admission his symptoms were controlled on non-steroidal anti-inflammatory drugs (NSAIDs) and prednisolone. He had no clinical signs of cardiac tamponade on presentation; however, a chest radiograph showed an enlarged cardiac silhouette and bilateral pleural effusions. A transthoracic echocardiogram and computed tomographic (CT) scan of the thorax (left panel) confirmed a large loculated pericardial effusion with compression of the right ventricular cavity. Aspiration of pleural fluid confirmed a transudate with a normal glucose. Pericardiocentesis resulted in an improvement in his symptoms. No malignant cells were seen and he was discharged for outpatient follow up. His symptoms recurred as did the pericardial effusion and he was transferred to a cardiothoracic centre for surgery. At operation a large and friable pericardial mass was debrided and a pericardial window was performed. Histology revealed fibrinous pericardium and chronic inflammatory infiltrates (middle and right panels). There was a further recurrence at four weeks and despite further drainage and aggressive immunosuppression he did not recover and the postmortem examination confirmed bilateral pulmonary emboli.

Pericardial compression in rheumatoid arthritis is rare. Pericardiocentesis and the formation of a pericardial window only provide temporary improvement and we suggest that the definitive treatment should be a pericardectomy. To our knowledge this is the only description of a histologically proven rheumatoid inflammatory mass presenting as cardiac compression.

P Bhat
B Chandrasekaran
E Barnes
badri53@hotmail.com



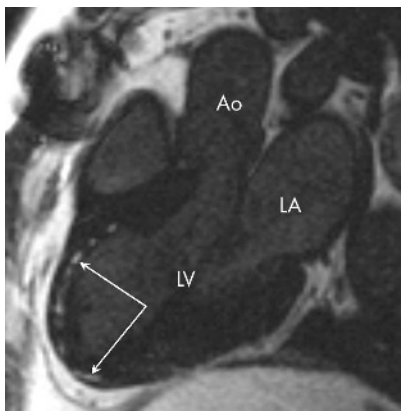
CT scan of the thorax showing a large pericardial collection compressing the right ventricle. There are also bilateral pleural effusions.



Haematoxylin and eosin stain showing fibrous pericardium containing large numbers of haemosiderin laden macrophages and a patchy chronic inflammatory infiltrate.

doi: 10.1136/hrt.2005.080952

Lipomatous metaplasia in myocardial infarction detected by cardiovascular magnetic resonance



Left ventricular outflow tract view; two-dimensional inversion recovery image before administration of contrast, arrows indicating high signal intensity in infarcted area. Ao, aorta; LA, left atrium; LV, left ventricle.

A 64-year-old man with a prior history of anterior wall myocardial infarction treated with thrombolysis in 1992, followed 10 years later by coronary artery bypass surgery, underwent cardiovascular magnetic resonance examination (CMR) for quantification of residual left ventricular function. Steady state free precession cine-CMR demonstrated thinned myocardium with dyskinesis of the distal anteroseptal and apical left ventricular regions consistent with an old myocardial infarction. Areas of unusual signal intensity compared to that of the surrounding myocardium were noted on cine-CMR within the infarcted region. These areas were confirmed to be composed of adipose tissue due to their characteristic high signal intensity on T1-weighted turbo spin echo images and complete disappearance of signal using fat suppression. The adipose tissue also had high signal intensity on two-dimensional inversion recovery images obtained before administration of gadolinium-based contrast (see panel) and was surrounded by areas of myocardial fibrosis demonstrated on late contrast-enhanced (LCE) images. These findings are consistent with lipomatous metaplasia in the region of an old myocardial infarction. This case demonstrates that a high signal intensity on LCE images may not always represent fibrosis alone in regions of old myocardial infarction.

R Nijveldt
C B Marcu
A C van Rossum
R.Nijveldt@vumc.nl